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Interim Procedures for Estimating Risks Associated with Exposures of Mixtures of Chlorinated Dibenzo-p-Dioxins and -Dibenzofurans (CDDs and CDFs)

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RISK ASSESSMENT FORUM

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Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-*p*-Dioxins and -Dibenzofurans (CDDs and CDFs)

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Preface

As part of its effort to address risks posed by chlorinated dibenzo-*p*-dioxins and chlorinated dibenzofurans (CDDs and CDFs) in the environment, the U.S. Environmental Protection Agency (EPA) has adopted an interim procedure, based on dioxin "toxicity equivalence" factors (TEFs), for estimating the hazard and dose-response of complex mixtures containing CDDs and CDFs in addition to 2,3,7,8-TCDD. The TEF procedure, and the scientific data upon which it is based, are the subject of this report.

This report, which has been extensively reviewed by EPA and external (non-EPA) experts, was prepared for EPA's Risk Assessment Forum (Forum) and was approved by the EPA Risk Assessment Council in August 1986. In September 1986, the report was reviewed by a special Subcommittee of the Agency's Science Advisory Board (SAB), a congressionally mandated body of independent scientists.

The SAB Subcommittee concurred with EPA's view that the TEF method is a reasonable interim approach to assessing the health risks associated with exposure to mixtures of CDDs and CDFs for risk management purposes. They noted that the method proposed may lack scientific validity and agreed with EPA on the importance of efforts to validate the method by selected experimental testing of hypotheses. The Agency received strong encouragement to continue research on other approaches to estimating risks for substances in mixtures. The Subcommittee also indicated that it was important that the interim approach be re-evaluated systematically by EPA as lessons are learned from toxicologic research and from application. Lastly, the group cautioned that the interim TEF method should be largely reserved for special situations where the components of the mixture are known, where the composition of the mixture is not expected to vary much with time, and where the extrapolations are consistent with existing animal data. Some aspects of the report have been revised to take the Subcommittee's comments into account.

These SAB comments reinforce EPA's views on the strengths and limitations of the TEF approach. Throughout development of the report, EPA scientists have emphasized that the TEF approach is an interim science policy to be used pending development of more rigorous and scientifically robust approaches, some of which are mentioned in the report. The Agency intends to encourage and to pursue a range of research activities which will both further test the hypotheses that underlie this interim procedure and lead to alternative, more direct approaches to determining the toxicity of CDD and CDF mixtures.

Research on CDDs and CDFs continues at a rapid pace, and the Agency is closely monitoring changes in the data base upon which the TEF approach has been established. Through an annual updating of the approach, the Forum will assure that TEF factors remain current with the existing animal data.

The TEF procedure will be used generally throughout the Agency for situations in which the components of the mixture are known (or can be reasonably anticipated) and where the composition is not expected to vary greatly with time.

On other issues the SAB Subcommittee and other peer reviewers recommended that EPA consider more explicitly the effects of pharmacodynamics (the bioavailability, absorption, distribution, metabolism, and elimination) of relevant environmental mixtures in whole animals when

assigning TEFs to the homologues and isomers of CDDs and CDFs. For example, studies suggest that higher chlorinated CDDs and CDFs are less likely to be absorbed during acute exposures. Further, some CDDs and CDFs are more likely to be metabolized and eliminated than are others. The Forum will review these issues and recommend changes in some TEFs, as approporiate.

In summary, the TEF approach provides a useful interim method for consistently interpreting the significance of CDD and CDF residues in the environment, until more direct methods are available. Users should be aware of the uncertainties associated with the procedure. In addition to the uncertainties inherent in the 2,3,7,8-TCDD quantitative risk assessment, which the TEF approach implicitly adopts, the approach includes the added qualitative assumption that the other CDDs and CDFs will demonstrate the same chronic effects as 2,3,7,8-TCDD. While there are good scientific reasons to expect this to be the case, the data to support this assumption are limited.

The Agency plans to update the TEFs on a regular basis, incorporating additional information as it becomes available so that the approach will reflect the best current scientific thinking. The intent is to replace this interim procedure with a more rigorous approach as research results permit.

I. Summary

The U.S. Environmental Protection Agency (EPA) is often confronted with the need to determine the risks associated with exposure to materials such as soot, incinerator fly ash, industrial wastes, and soils which contain complex mixtures of chlorinated dibenzo-*p*-dioxins (CDDs) and chlorinated dibenzofurans (CDFs).[1] Recognizing the public and toxicological concern generated by these chemicals and the significant gaps in our ability to evaluate the human health potential of these compounds by existing procedures, the CDD/CDF Technical Panel of the Risk Assessment Forum (Forum) is recommending an interim method to aid in the assessment of the human health risks posed by mixtures of CDDs and CDFs until data gaps are filled.

The Technical Panel has reviewed a spectrum of approaches for making such assessments, consistent with EPA's Guidelines for the Health Risk Assessment of Chemical Mixtures, and has concluded that a direct biological assessment of the toxicity of complex mixtures of CDDs and CDFs is preferred. However, a validated bioassay that can plausibly be applied to such mixtures is not now available, although promising research is in progress in the area. An alternative approach involves explicit analysis and toxicological determination of each of the constituent CDD/CDF congeners. The data required for such an approach also need to be developed and are not likely to be generated soon. The Forum therefore concludes that, as an interim science policy measure, a reasonable estimate of the toxic risks associated with a mixture of CDDs and CDFs can be made by taking into account the distribution of CDD/CDF congeners or homologues and the likely relative toxicity of these compounds. This document describes the recommended interim procedure for generating the "2378-TCDD equivalence" of complex mixtures of CDDs and CDFs, based on congener- or homologue-specific data, and for using such information in assessing risk. (The recommendations are summarized in the rightmost column of Table 1.)

The Forum acknowledges that this procedure is not based on a thoroughly established scientific foundation. Instead, the approach represents a consensus recommendation for interim science policy, subject to change as additional data are available. The approach is judged to be applicable to mixtures of CDDs and CDFs, but should not be construed as being applicable as well to mixtures of other chemicals.

The basis of this approach, i.e., the assignment of toxicity equivalence factors (TEFs) is subject to revision as new scientific data become available in the future. Consequently, risk assessors and risk managers are urged to use informed discretion, noting specific problems on a case-by-case basis, when applying the procedure to any particular situation. The Forum urges the support of research to test further the hypotheses that underlie this interim procedure and to develop the preferred approaches.

[1]See Appendix A for the nomenclature and conventions used in this paper.

Table 1. Some Approaches to Estimating Relative Toxicities of PCDDs and PCDFs

Basis/ compound	Swiss[a]	Grant[b] Olie[c] Commoner[d]	New York State[e]	Ontario[f]	FDA[g]	CA[h]	EPA[i] 1981	EPA current recommend.
(Basis)	Enzyme		LD_{50}	Various effects	Various effects			Various effects
Mono thru di CDDs	0	0	0	0	0	0	0	0
Tri CDDs	0	0	0	1	0	0	0	0
2378-TCDD	1	1	1	1	1	1	1	1
other TCDDs	0.01	1	0	0.01	0	0	1	0.01
2378-PeCDDs	0.1	0.1	1	1	0	1	0	0.5
other PeCDDs	0.1	0.1	0	0.01	0	0	0	0.005
2378-HxCDDs	0.1	0.1	0.03	1	0.02	1	0	0.04
other HxCDDs	0.1	0.1	0	0.01	0.02	0	0	0.0004
2378-HpCDDs	0.01	0.1	0	1	0.005	1	0	0.001
other HpCDDs	0.01	0.1	0	0.01	0.005	0	0	0.00001
OCDD	0	0	0	0	<0.00001	1	0	0
2378-TCDFs	0.1	0.1	0.33	0.02	0	1	0	0.1
other TCDFs	0.1	0.1	0	0.0002	0	0	0	0.001
2378-PeCDFs	0.1	0.1	0.33	0.02	0	1	0	0.1
other PeCDFs	0.1	0.1	0	0.0002	0	0	0	0.001

Table 1. *(continued)*

Basis/ compound	Swiss[a]	Grant[b] Olie[c] Commoner[d]	New York State[e]	Ontario[f]	FDA[g]	CA[h]	EPA[i] 1981	EPA current recommend.
(Basis)	Enzyme		LD_{50}	Various effects	Various effects			Various effects
2378-HxCDFs	0.1	0.1	0.01	0.02	0	1	0	0.01
other HxCDFs	0.1	0.1	0	0.0002	0	0	0	0.0001
2378-HpCDFs	0.1	0.1	0	0.02	0	1	0	0.001
other HpCDFs	0	0.1	0	0.0002	0	0	0	0.00001
OCDF	0	0	0	0	0	0	0	0

[a]Swiss Government, 1982.
[b]Grant, 1977.
[c]Olie et al., 1983.
[d]Commoner et al., 1984.
[e]Eadon et al., 1982.
[f]Ontario, 1982.
[g]U.S. DHHS, 1983.
[h]Gravitz et al., 1983.
[i]U.S. EPA, 1981.

II. The Need for a Procedure for Assessing the Risk Associated with Exposure to Complex Mixtures of CDDs and CDFs

During the late 1970s, the Agency was faced with assessing the human health significance of exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD). In preparation for the cancellation hearings for the herbicides 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and Silvex, the Agency generated risk assessments for several toxic responses for 2,3,7,8-TCDD. The quantitative cancer risk assessment developed by the Carcinogen Assessment Group was later adapted for use in the Water Quality Criteria (WQC) Document for 2,3,7,8-TCDD (U.S. EPA, 1984a). In addition to carcinogenicity concerns, the WQC document contains an assessment of systemic toxicity based on reproductive effects resulting from exposure to 2,3,7,8-TCDD.

Later, it became clear that exposure situations exist in the country which involve more than 2,3,7,8-TCDD alone. Data on emissions from combustion sources (e.g., hazardous waste and municipal waste incinerators) and contents of waste from certain industrial production processes indicate that the majority of the 75 CDDs and 135 CDFs can be detected in the environment.

In recent years, the reporting of at least homologue-specific data for the CDDs and CDFs has become commonplace, and the Agency has taken some steps to address the significance of these findings. For example, the Health Assessment Document for Polychlorinated Dibenzo-*p*-Dioxins, prepared for the Office of Air Quality Planning and Standards (U.S. EPA, 1985b), contains a quantitative risk assessment for a mixture of hexachlorodibenzo-*p*-dioxins (HxCDDs) based on carcinogenicity studies conducted by the National Cancer Institute. These concerns have also led to regulatory action; e.g., several industrial wastes containing tetra-, penta-, and hexa-chlorodioxins, and -dibenzofurans were recently designated by the Agency as EPA hazardous wastes.

Faced with increasing amounts of isomer- and homologue-specific data, and recognizing the significant potency and structure-activity relationships exhibited in *in vivo* and *in vitro* studies of CDDs and CDFs, the Technical Panel perceives a need to address more generally the potential risks posed by the congeners other than 2,3,7,8-TCDD and the mixture of HxCDDs.[2] Detailed consideration of the toxicity of the vast majority of the CDDs and CDFs is limited by the lack of a complete toxicological data base on most of the congeners. Further, it is unlikely that many long-term test results will be availabale soon. For example, research on 2,3,7,8-TCDD has been under way for more than two decades at an estimated cost of more than one hundred million dollars. Although this chemical has been investigated to a much greater extent than any of the other CDDs and CDFs, unanswered questions remain. Therefore, the Forum believes that an interim science policy position should be adopted for use in assessing risks associated with CDD/CDF mixtures, until more definitive scientific data are available.

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[2]In the early 1980s, the Agency developed a method for an approximate assessment of the risks of the emission of CDDs and CDFs associated with the high-temperature incineration of PCBs and combustion of municipal waste (U.S. EPA, 1981; U.S. EPA, 1982); see Table 1. The procedure presented in this document is a refinement of that approach. A comparison of a variety of methods is included in Appendix B.

III. Approaches to Hazard Assessment for CDD/CDF Mixtures

A. The Ideal Approach—Long-Term, Whole-Animal Toxicity Assay of Mixtures

Under ideal conditions, an assessment of the toxicity of a mixture of chemicals is best accomplished by direct evaluation of its toxic effects, e.g., by determining the effects of chronic exposure in an experimental animal (U.S. EPA, 1985a). Such an assessment is time-consuming and costly and would theoretically have to be performed for each of the many mixtures of environmental importance. Therefore, this idealized approach would cause unacceptable delays in addressing the potential health risks associated with exposures to CDD/CDF mixtures.

Long-term animal studies might be considered for some categories of CDD/CDF sources which have characteristic compositions; e.g., emission from some combustion sources. However, the need for an interim approach would remain.

B. A Promising Approach—Short-Term, Biological Assay of Mixtures

An alternative, and perhaps more achievable, approach to hazard assessment of a mixture is a short-term assay (*in vivo* or *in vitro*) that indirectly provides a measure of the mixture's potential toxicity. In the case of mixtures containing CDDs and CDFs, short-term assays are under development that directly determine the 2,3,7,8-TCDD-like response which could be used as a *measure of the toxicity of the mixture as a whole.* Such assays take advantage of the similar toxic end points induced by CDDs and CDFs, and have been used to assess the potential health hazards of exposure to CDD/CDF-contaminated soot from PCB fires (Eadon et al., 1982; Gierthy and Crane, 1984; Gravitz et al., 1983), and for predicting the potential toxicity of incinerator fly ash (Rizzardini et al., 1983; Sawyer et al., 1983).

Although the development of such "mixture assays" is progressing rapidly (e.g., Safe et al., 1985), additional work is required to more fully validate the assay findings for specific toxic end points, especially chronic effects, and aspects of pharmacokinetics need to be considered. The Forum, recognizing the importance of short-term assays, encourages research in this area.

C. A Reductionist Approach—Additivity of Toxicity of Components

In the absence of a fully developed "mixture assay," the components in a mixture of CDDs and CDFs could theoretically be identified and quantified by analytical chemists. Then the toxicity of the mixture could be estimated by *adding the toxicity contributed by each of its components.* In the case of most environmental mixtures, however, this method would be of limited value since congener-specific analyses for the 75 CDDs and 135 CDFs potentialy present in the mixture are seldom available. In addition, there

is little informmation available on the toxic potency of most of these congeners. Therefore, this approach is not viable at this time; nor is it likely to be feasible in the near future.

D. An Interim Approach—2378-TCDD Toxicity Equivalence Factors (TEFs)

The Forum recommends a fourth alternative for estimating the risks associated with exposure to complex mixtures of CDDs and CDFs. In this approach, as in approach C above, information is obtained on the concentrations of homologues and/or congeners present in the mixture. Then, using the available toxicological data and reasoning on the basis of structure-activity relations, the significance of the exposure to each of the components is estimated and expressed as an "equivalent amount of 2378-TCDD." Combining this information with hazard information on 2,3,7,8-TCDD, and assuming additivity of effects, the risks associated with the mixture of CDDs and CDFs can be estimated if exposure is known. Key to the approach are the 2378-TCDD Toxicity Equivalence Factors (TEFs) which are derived in Section IV.

The general approach using TEFs as outlined here is not unique; several organizations have used similar approaches (see Table 1).

At one extreme, all CDDs and CDFs could be assumed to be as toxic as 2,3,7,8-TCDD (all TEFs = 1). This position is not recommended since the limited long-term data (2-year cancer bioassays) on 2,3,7,8-TCDD and a mixture of 2378-HxCCDs (and the greater body of short-term data on many CDDs and CDFs) indicate that such an assumption is overly conservative. At the other extreme one could totally ignore the presence of CDDs and CDFs other than those for which adequate long-term data are available (most TEFs = 0). This position is not recommended in light of the similar toxic properties of several of these compounds and the structure-activity relationship demonstrated for effects resulting from less than lifetime exposures.

Instead, the Forum recommends that the TEF procedure presented in Section IV be adopted as a matter of science policy on an interim basis, subject to revision as new experimental data become available. Based on the available scientific information, the Forum believes that this approach represents an appropriate means of approximating the potential risk of exposure to mixtures of CDDs and CDFs for purposes of risk management.

The approach will enable the Agency to deal with many, but not all, of its problems; e.g., assigning priority to Superfund sites, estimating the extent to which a hazardous waste site should be cleaned up, guiding decisions on which manufacturing wastes can be delisted as EPA hazardous wastes, and estimating risks associated with the emission of CDDs and CDFs from combustion sources.

The remainder of this document discusses the TEF approach in greater detail, illustrates its use in risk assessment, and identifies additional research, the results of which would provide information for adjustments to this interim approach.

IV. The 2378-TCDD Toxicity Equivalence Factors (TEFs) Approach to Assessing the Toxicity of Complex Mixtures of CDDs and CDFs

2,3,7,8-TCDD is one of 75 CDDs. Exceptionally low doses of this compound elicit a wide range of toxic responses in many animals, e.g., adverse reproductive effects, thymic atrophy, and a "wasting syndrome" leading to death. Although the Agency prefers definitive human evidence when assessing the potential human carcinogenicity of chemicals, such data are rarely available and are lacking in the case of CDDs and CDFs period. However, EPA's Carcinogen Assessment Group (CAG) has determined that, based on demonstrated effects in animals, there is sufficient evidence to regard 2,3,7,8-TCDD and a mixture of two 2378-HxCDDs as probable human carcinogens. The CAG quantitative assessment indicates that these chemicals are among the most potent animal carcinogens evaluated by the Agency to date. Limited data suggest that some of the other CDDs may have other toxic effects similar to those of 2,3,7,8-TCDD, again at very low doses.

Moreover, these toxicity concerns are not restricted to CDDs. Limited experimental data, supplemented by structure/activity relationships in *in vitro* tests that are correlated with *in vivo* toxic effects of CDFs, indicate that some of these compounds exhibit "2,3,7,8-TCDD-like" toxicity (Bandiera et al., 1984; Okey et al., 1984; Safe et al, 1985).

The biochemical mechanisms leading to the toxic response resulting from exposure to CDDs and CDFs are not known in detail. However, experimental data have accumulated which suggest that an important role in the development of systemic toxicity resulting from exposure to these chemicals is played by an intracellular protein, the Ah receptor, the putative product of a gene locus designated Ah. This receptor binds halogenated polycyclic aromatic molecules, including CDDs and CDFs. It has been postulated that the Ah locus controls several pleiotropic responses: a limited, but widely expressed gene complex that includes the structural genes for aryl hydrocarbon hydroxylase (AHH) expression, and, in a few organs, such as skin and thymus, a second gene complex regulating cell proliferation and differentiation (Knutson and Poland, 1980; Neal et al., 1982; Greenlee et al.., 1985a).

In several mouse strains, the expression of toxicity of 2,3,7,8-TCDD-related compounds, including cleft palate formation, liver damage, effects on body weight gain, thymic involution, and chloracnegenic response, has been correlated with their binding affinity for the Ah receptor, and with their ability to induce several enzyme systems, some of which have been linked to the expression of carcinogenicity (Poland and Knutson, 1982; Bandiera et al.,1984; Madhukar et al., 1984; Poland et al., 1985; Safe et al., 1985; Vickers et al., 1985). Structure-activity studies also link the enhanced *in vitro* cell differentiation caused by these compounds to the presence of the Ah receptor (Greenlee et al., 1985b).

However, it has also been noted that the cytosolic receptor binding alone may not be the sole determinant of the capacity for AHH induction (Neal, 1985; Okey and Vella, 1984). In interspecies comparisons there are poor correlations between the concentration of cellular Ah receptor, its ability to bind 2,3,7,8-TCDD and AHH induction (Denison and Wilkinson, 1985; Gasiewicz and Rucci, 1984; Neal, 1985); and in the mouse the development

of TCDD-induced liver toxicity cannot be ascribed solely to the presence of the Ah receptor (Greig et al., 1984).

A recent review concludes that although there are inconsistencies across species in the Ah receptor's being the sole mechanism of toxicity of CDDs and CDFs, the data suggest that the binding of these compounds to the receptor is in some way related to some of the biological effects seen in experimental animals (Neal, 1985).

Table 2 summarizes information on a variety of end points elicited by CDDs and CDFs: acute toxicity, carcinogenicity, reproductive effects, receptor, binding, enzyme induction, and *in vitro* cell transformation. For ease of comparison, the data are normalized to unity for 2,3,7,8-TCDD. For example, 2378-HxCDDs have about 5% the Ah receptor binding strength of 2,3,7,8-TCDD. Their reproductive toxicity and carcinogenic potency are, respectively, about 1% and 4% that of 2,3,7,8-TCDD. Kociba and Cabey (1985) recently presented similar data.

The structure/activity generalizations based on the data in Table 2 support the generalizations in the literature concerning the congeners that are most likely to be of toxic concern (Poland and Knutson, 1982; Gasiewicz and Rucci, 1984; Bandiera et al., 1984). That is, congeners that are substituted in the lateral 2,3,7 and 8 positions are likely to exhibit toxic effects at lower doses than other congeners. This includes the 15 tetra-, penta-, hexa- and heptachlorinated CDDs and CDFs listed in Table 3.[3]

The "2378-TCDD equivalence factors" (TEFs) listed in Tables 1 and 3 were assigned using several criteria.

1. Definitive data on human carcinogenicity.
2. In the absence of definitive data on human carcinogenicity, information on carcinogenic potency is based on long-term animal studies which takes precedence over any other data.
3. When carcinogenic activity has not been demonstrated, data on reproductive effects become determinative because of the significance of this end point in humans. In addition, the estimated exposure levels potentially resulting in reproductive and carcinogenic effects are similar.
4. When neither carcinogenic nor reproductive effects have been demonstrated, the weight of the evidence of the *in vitro* test data is estimated. To simplify the approach and to acknowledge the approximate nature of the approach, these estimates are rounded off to the nearest order of magnitude. Somewhat more weight is placed on data from receptor binding interaction and oxidative enzyme

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[3]The Technical Panel is aware that some investigators (e.g., Grant, 1977; Olie et al., 1983; Commoner et al., 1984; and Ontario, 1982, 1984) have broadly defined congeners of concern to include those tri- to hepta- congeners which are substituted with at least three chlorines in the four lateral (2, 3, 7, and 8) positons. The toxicity data (Table 2) do not strongly support this extended range of concern. Further the increased level of complexity invoked by including these additional congeners suggests a greater level of accuracy and resolution than the Technical Panel believes is presently warranted by the TEF approach.

The Technical Panel is also aware that receptor binding data suggest a relatively high potential toxicity for 1,2,4,6,7-PeCDF. Examination of stereochemical models shows that the 4 and 6 positions of CDFs exhibit partial overlap with the lateral chlorine groups of 2,3,7,8-TCDD (Bandiera et al., 1984). However, this increased receptor binding activity is not reflected in an increased potency of 1,2,4,6,7-PeCDF as an enzyme inducer (see Table 2), an end point which has been shown to correlate with subchronic toxicity (Safe et al., 1985). Therefore, the Technical Panel is treating 1,2,4,6,7-PeCDF as a "non-2378-congener" at this time; however, additional data could lead to a change in this position.

1,2,3,6,7- and 2,3,4,6,7-PeCDF are almost as potent as 2378-PeCDF in the induction of AHH activity in human lymphoblastoid cells *in vitro* (see Table 2). However, because this assay seems to yield relative potencies that do not agree with other short-term tests, and because dose-response data are not available for this assay, these data are not included in the overall evaluation at the present time.

Table 2. Potencies of Dioxins Relative to 2,3,7,8-TCDD

Chemical	Guinea pig LD_{50}	Carcino-genicity	Reproductive/ teratogenic effects	Receptor binding	Enzyme Induction: AHH: Animal cells	Enzyme Induction: AHH: Human cells	Enzyme Induction: EROD	Cell keratin.	Flat (XB) cell assay	Immuno-toxicity in vitro
CDDs:										
Mono thru tri	$<10^{-4}$[e]	--	--	.001-.01[e]	<.001[f]	--	--	.01[e]	--	.005[p]
2378-TCDD	1[a]	1[b]	1[c,i]	1[e]	1[e]	1[m]	1[g]	1[e]	1[i]	1[o,p]
TCDDs	<.001[a]	--	<.001[k]	<.01-.16[e]	<.001-.02[g]	--	--	<.001-.01[e]	--	--
2378-PeCDD	.67[a]	--	--	1[e]	.02-.2[g]	--	--	.5[e]	--	--
PeCDDs	.002[a]	--	--	--	<.001[g]	--	--	--	--	--
2378-HxCDDs	.03[a]	.04[b]	.01[c]	.05[e]	.001-.1[g]	--	--	.005[e]	--	--
HxCDDs	--	--	--	--	<.001[g]	--	--	--	--	--
2378-HpCDDs	.004[a]	--	--	--	.002-.004[g,f]	--	--	--	--	--
HpCDDs	.002[a]	--	--	--	<.001[f]	--	--	--	--	--
OCDD	--	--	<.00001[k]	--	<.001[f]	--	--	--	--	--
CDFs:										
Mono thru tri	--	--	--	≤.001-.02[d,h]	<.001[d]	<.001[d]	--	.001[e]	--	--
2378-TCDF	.28; .5[a]	--	.03-.13[j,k]	.3[e]; .24[h]; .4[l]	.01-.4[f,h,m]	.4[m]	.1[h]	.05[e]	.1[i]	.1[o], 1[p]

Table 2. *(continued)*

Chemical	Guinea pig LD_{50}	Carcinogenicity	Reproductive/ teratogenic effects	Receptor binding	Enzyme Induction: AHH, Animal cells	Enzyme Induction: AHH, Human cells	Enzyme Induction: EROD	Cell keratin.	Flat (XB) cell assay	Immunotoxicity in vitro
TCDFs	--	--	--	.001-.05[d,e]	≤.001[d]; .04[m]	.4[m]	≤.005[d]	--	--	--
2378-PeCDF	--	--	--	.13[d]; .7[e]; .6[h]	<.3[d]; .4[m]	.8[m]	.1[d]	--	--	--
12467-PeCDF	--	--	--	.15[h]	.002[h]	--	<.001[h]	--	--	--
PeCDFs	--	--	--	.001-.1[d,e]	≤.001-2[d,n,m]	.6[m]	≤.001[h]	--	--	--
2378-HxCDFs	.017[a]	--	--	.04-.5[e,h]	.05-.2[h,m]	.9[m]	.1-.5[h]	--	--	--
HxCDFs	--	--	--	.001[e,h]	.001[m];.002[h]	--	.006[h]	--	--	--
2378-HpCDFs	--	--	--	--	.004[g]	--	--	--	--	--
HpCDFs	--	--	--	<.001[h]	<.001[f]	--	--	--	--	--

[a] McKinney and McConnell, 1982; Moore et al., 1979.
[b] U.S. EPA, 1984a.
[c] Murray et al., 1979; Schwetz et al., 1973; Weber et al., 1984.
[d] Bandiera et al., 1983.
[e] Knutson and Poland, 1980.
[f] Bradlaw et al., 1979.
[g] Bradlaw et al., 1980.
[h] Bandiera et al., 1984.
[i] Hassoun et al., 1984.
[j] Gierthy and Crane, 1985.
[k] Weber et al., 1984.
[l] Poland et al., 1979.
[m] Nagayama et al., 1985a,b.
[n] Poland et al., 1976.
[o] Dencker et al., 1985.
[p] Greenlee et al., 1985b.

Table 3. ***CDD/CDF Isomers of Most Toxic Concern[a]***

Dioxin		*Dibenzofuran*	
Isomer	*TEF[b]*	*Isomer*	*TEF[b]*
2,3,7,8-TCDD	*1*	*2,3,7,8-TCDF*	*0.1*
1,2,3,7,8-PeCDD	*0.5*	*1,2,3,7,8-PeCDF*	*0.1*
		2,3,4,7,8-PeCDF	*0.1*
1,2,3,4,7,8-HxCDD	*0.04*	*1,2,3,4,7,8-HxCDF*	*0.01*
1,2,3,7,8,9-HxCDD	*0.04*	*1,2,3,7,8,9-HxCDF*	*0.01*
1,2,3,6,7,8-HxCDD	*0.04*	*1,2,3,6,7,8-HxCDF*	*0.01*
		2,3,4,6,7,8-HxCDF	*0.01*
1,2,3,4,6,7,8-HpCDD	*0.001*	*1,2,3,4,6,7,8-HpCDF*	*0.001*
		1,2,3,4,7,8,9-HpCDF	*0.001*

[a]In each homologous group, the relative toxicity factor for the isomers not listed above is 1/100 of the value listed above.

[b]TEF = Toxicity Equivalence Factor = relative toxicity assigned.

induction, due to the correlations between these *in vitro* end points and certain *in vivo* systemic efforts; e.g., thymic atrophy and body weight loss.

The above criteria were applied as described below.

1. Since the primary concern is with chronic effects, the relative carcinogenicity response (Table 2) for 2,3,7,8-TCDD and the mixture of two 2378-HxCDDs[4] were used to generate the TEF for 2378-PeCDD. The TEF for 2378-PeCDD (0.5) is the arithmetic mean of the carcinogenic potency values for 2,3,7,8-TCDD (1) and 2378-HxCDDs (0.04). Data on receptor binding, enzyme induction, and cell keratinization generally support this values.
2. 2,3,7,8-TCDF is assigned a TEF of 0.1 primarily because it is 1 to 2 orders of magnitude (OMs) less potent than 2,3,7,8-TCDD in reproductive toxicity tests. Also, it is about one OM less potent than 2,3,7,8-TCDD in the *in vitro* tests.
3. The 2378-PeCDF congeners are assigned a TEF of 0.1 due to the responses seen in *in vitro* tests. Greater reliance was placed on the animal enzyme induction studies due to the more significant correlations observed between this end point and subchronic responses than have been observed with the receptor binding end point. The human cell data were accorded less weight because these experiments were conducted at only one exposure concentration.
4. Because *in vitro* data in general show HxCDFs to be about one tenth as potent as PeCDFs, their TEF is assigned a value of 0.01 (0.1/10). Further, the data generally suggest that CDFs are somewhat less toxic than the analogous CDDs. Therefore, the TEF for 2378-HxCDFs should be less than that of the 2378-HxCDDs (0.04).
5. The 2378-HpCDDs and 2378-HpCDFs are assigned TEFs 3 OM less than that for 2,3,7,8-TCDD because the enzyme induction potencies of these congeners differ from that of 2,3,7,8-TCDD by about this factor.

[4]See Appendix A, item 6, for explanation of notation.

6. Based on the data in Table 2, the non-2378-substituted isomers are 1 to 2 OMs less potent than the 2378-substituted isomers. Since these data are limited to *in vitro* systems, a factor of 0.01 is applied to the non-2378-substituted, as compared to the 2378-substituted congeners.

With the exception of 2,3,7,8-TCDD, the 2378-HxCDDs, and 2378-TCDF, the TEFs are not based on the results of major animal (reproductive, carcinogenic) studies. Generally, TEFs are based on estimates of the relative toxicity in *in vitro* tests whose relationship to the chronic effects of concern is largely presumptive. However, as discussed above, studies on systemic effects continue to reinforce the view that the short-term assays provide important fundamental information on the toxicity of the CDDs and CDFs.

In summary, the Forum concludes that there is a sufficiently plausible basis for the TEF approach of estimating risks associated with exposures to CDDs and CDFs and recommends that the Agency adopt the approach, on an interim basis, as a matter of science policy. The TEFs should be revised as additional scientific information is developed. It should be noted that this general approach to estimating such CDD/CDF risks has been taken by other regulatory groups (see Table 1 and Appendix B).

V. Applications to Risk Assessment

In general, as assessment of the human health risk of a mixture of CDDs and CDFs, using the TEF approach, involves the following steps:

1. Analytical determination of the CDDs and CDFs in the sample.
2. Multiplication of congener concentrations in the sample by the TEFs in Table 1 to express the concentration in terms of 2378-TCDD equivalents.
3. Summation of the products in step 2 to obtain the total 2378-TCDD equivalents in the sample.
4. Determination of human exposure to the mixture in question, expressed in terms of 2378-TCDD equivalents.
5. Combination of exposure from step 4 with toxicity information on 2,3,7,8-TCDD (usually carcinogenicity and/or reproductive effects) to estimate risks associated with the mixture.

In cases in which the concentrations of the 15 congeners are known:

$$\text{2378-TCDD Equivalents} = \Sigma\,(\text{TEF of each 2378-CDD/CDF congener} \times \text{the concentration of the respective congener}) + \Sigma\,(\text{TEF of each non-2378-CDD/CDF congener} \times \text{the concentration of the respective congener})$$

Samples of this calculation for several environmental mixtures are provided in Table 4.

In cases where only the concentration of homologous groups is known, i.e., no isomer-specific data are available, different approaches are possible. For example, the assumption that the 2378-congeners of concern constitute all of the CDDs and CDFs present in the mixture is likely to provide an upper-bound, most conservative estimate of the toxicity. Alternatively, one could assume that the occurrence of each of the congeners in the mixture has equal probability (Olie et al., 1983; Commoner et al., 1984). For instance 2,3,7,8-TCDD is one of 22 possible TCDDs and would constitute about 4% of a mixture of isomers occurring with equal probability. In other situations particular knowledge of chemical reaction parameters, process conditions, and results from related studies (e.g., congener distributions in emissions form combustion sources) might enable one to estimate the relative occurrence of 2378-congeners. However, one must be careful to explicitly explain and justify whatever assumptions are made. Table 5 illustrates the results obtained using different methods to estimate the proportion of 2378 to non-2378 isomers in the absence of analytical data for individual isomers.

The calculated 2378-TCDD equivalents can then be used to assess the health risk of a mixture. As an explicit example, consider a municipal solid waste (MSW) combustor whose particulate emissions, the CDD/CDF mixture in question, are the same as the electrostatic precipitator (ESP) catch cited in columns 5 and 6 of Table 4. The sample is estimated to contain 32 ppb 2378-TCDD equivalents; i.e., 32 picograms of 2378-TCDD equivalents per milligram of mixture. Suppose that an exposure analysis indicates that a person living downwind from the incinerator receives an average daily dose of 1 ng of the mixture/kg body weight resulting from inhalation (i.e., without consideration of other possible routes of exposure). This exposure estimate is combined with the upper-bound carcinogenic potency of 2,3,7,8-TCDD (1.6×10^5 per mg/kg-day [U.S. EPA, 1984b]) to generate the upper 95%

Table 4. PCDDs/PCDFs in Some Environmental Samples

		Air partics. St. Louis[a]		MSW ESP dust[a]		Lake sediment[a]		1982 Milorganite[d]		MSW fly ash[f] Ontario		MSW fly ash[f] Oslo	
Isomer	TEF	CDD/F conc. (ppb)	TCDD eqts.	CDD/F conc. (ppb)	TCDD eqts.	CDD/F conc. (ppb)	TCDD eqts.	CDD/F conc. (ppt)	TCDD eqts.	CDD/F conc. (ppt)	TCDD eqts.	CDD/F conc. (ppt)	TCDD eqts.
TCDDs	1	0.2	0.2	5	5	0	0	206	206	541	541	ND	--
PeCDDs	0.5	1	0.5	10	5	0.1	0.05	--	--	467	234	11	5.5
HxCDDs	0.04	1.2	0.048	160	6.4	0.34	0.014	2768	110.7	591	24	51	2
HpCDDs	0.001	25	0.025	120	0.12	0.5	0.001	7600	7.6	434	0.43	119	0.12
OCDD	0	170	0	260	0	1.3	0	60000	0	467	0	186	0
TCDFs	0.1	--	--	40	4	0.13	0.013	--	--	--	--	--	--
PeCDFs	0.1	--	--	80	8	0.14	0.014	--	--	--	--	--	--
HxCDFs	0.01	--	--	280	2.8	0.38	0.004	--	--	--	--	--	--
HpCDFs	0.001	--	--	160	0.16	1.13	0.001	--	--	--	--	--	--
OCDF	0	--	--	40	0	0.14	0	--	--	--	--	--	--
Total TCDD eqts.			0.08		32		0.10		324		799		7.3

Table 4. *(continued)*

Isomer	TEF	Thermal degradation prods. from dielectric fluids[a]				Japanese MSW[b]				Commercial CPs				Soot from PCB fire[g]	
		Run 8-13-40		Run 8-30-61 ASKL		Pt. A TEF		Pt. B TEF		246TCP[c]		PCP[c]			
		CDD/F conc. (ng)	TCDD eqts.	CDD/F conc. (μg)	TCDD eqts.	CDD/F conc.	TCDD eqts.	CDD/F conc.	TCDD eqts.	CDD/F conc. (ppm)	TCDD eqts.	CDD/F conc. (ppm)	TCDD eqts.	CDD/F conc. (ppm)	TCDD eqts.
						[lb/MMBTU($\times 10^{-6}$)]									
TCDDs															
2378	1	0	0	0	0	0.1	0.1	0.58	0.58	<0.1	--	<0.1	--	0.6	0.6
other	0.01													0.6	0.01
PeCDDs															
2378	0.5	0	0	0	0	0.07	0.035	0.47	0.24	<0.1	--	<0.1	--	2.5	1.25
other	0.002													2.5	0.01
HxCDDs															
2378	0.04	0	0	0	0	0.04	0.002	0.36	0.014	<1	--	2.5	0.1	1.1	0.04
other	0.0004													3.6	--
HpCDDs															
2378	0.001	0	0	330	0.33	0.02	<0.001	0.08	<0.001	<1	--	175	0.18	3	--
other	0.00001													4	--
OCDD	0	0	0	37	0	0.01	0	0.04	0	<1	0	500	0	2	0

Table 4. *(continued)*

Isomer	TEF	Thermal degradation prods. from dielectric fluids[a] Run 8-13-40 CDD/F conc. (ng)	Run 8-13-40 TCDD eqts. (ng)	Run 8-30-61 ASKL CDD/F conc. (μg)	Run 8-30-61 ASKL TCDD eqts. (μg)	Japanese MSW[b] Pt. A TEF CDD/F conc. [lb/MMBTU(10^{-6})]	Pt. A TEF TCDD eqts. [lb/MMBTU(10^{-6})]	Pt. B TEF CDD/F conc. [lb/MMBTU(10^{-6})]	Pt. B TEF TCDD eqts. [lb/MMBTU(10^{-6})]	Commercial CPs 246TCP[c] CDD/F conc. (ppm)	246TCP[c] TCDD eqts. (ppm)	PCP[c] CDD/F conc. (ppm)	PCP[c] TCDD eqts. (ppm)	Soot from PCB fire[g] CDD/F conc. (ppm)	Soot from PCB fire[g] TCDD eqts. (ppm)
TCDFs															
2378	0.1	690	69	1400	140	1.31	0.131	1.25	0.125	1.5	0.15	<0.1	--	12	1.2
other	0.001													16	0.01
PeCDFs															
2378	0.1	43	4.3	6400	640	0.38	0.038	0.46	0.046	17.5	1.75	<0.1	--	358	35.8
other	0.001													312	0.3
HxCDFs															
2378	0.01	7	0.07	910	9.1	0.06	0.006	0.06	0.006	36	3.6	<0.3	--	670	6.7
other	0.0001													295	0.03
HpCDFs															
2378	0.001	0	0	29	0.029	0.01	<.001	0.02	<.001	4.8	0.005	19	0.019	285	0.29
other	0.00001													172	0
OCDF	0	0	0	3.4	0	0.004	0	0.01	0	<1	0	25	0	40	0
Total TCDD eqts.			73		789		0.3		1.02		5.5		0.3		46

[a] U.S. EPA, 1984c.
[b] Cooper Engineers, 1984.
[c] Rappe, 1984
[d] Lamparski et al., 1984.
[e] Czuwa and Hites, 1984.
[f] Tong et al., 1984.
[g] Des Rosiers, 1984.

Table 5. Use of the TEF Approach

Isomer	TEF	Propn. factor	PCB fire soot[a] CDD/F conc. (ppm)	PCB fire soot[a] TCDD eqts. (ppm) A[c]	B[c]	C[c]	D[c]	MSW fly ash[b] Sample 1 CDD/F conc. (ppb)	Sample 1 TCDD eqts. (ppb) A[c]	B[c]	D[c]	Sample 2 CDD/F conc. (ppb)	Sample 2 TCDD eqts. (ppb) A[c]	B[c]	D[c]
Total TCDDs	1	1	1.2	1.2				85	85			2.7	2.7		
2378 TCDDs	1	0.05	1.2		0.2	0.6		85		4.3		2.7		0.1	
other TCDDs	0.01	0.95	1.2		--[d]	--		85		0.8		2.7		--	
Total PeCDDs	0.5	1	5.0	2.5				213	107			6.6	3.3		
2378 PeCDDs	0.5	0.07	5.0		0.2	1.3		213		7.0		6.6		0.2	
other PeCDDs	0.005	0.93	5.0		--	--		213		1.0		6.6		--	
Total HxCDDs	0.04	1	4.7	0.2				354	14.2			11.6	0.5		
2378 HxCDDs	0.04	0.3	4.7		0.1	--		354		4.3		11.6		0.1	
other HxCDDs	0.0004	0.7	4.7		--	--		354		0.1		11.6		--	
Total HpCDDs	0.001	1	7	--		--		184	0.2			5.7	--		
2378 HpCDDs	0.001	0.5	7		--			184		0.1		5.7		--	
other HpCDDs	0.00001	0.5	7		--			184		--		5.7		--	
Total TCDFs	0.1	1	28	2.8				209	20.9			7.0	0.7		
2378 TCDFs	0.1	0.03	28		0.1	1.2		209		0.6		7.0		--	
other TCDFs	0.001	0.97	28		--	--		209		0.2		7.0		--	

Table 5. *(continued)*

Isomer	TEF	Propn. factor	PCB fire soot[a] CDD/F conc. (ppm)	PCB fire soot[a] TCDD eqts. (ppm) A[c]	B[c]	C[c]	D[c]	MSW fly ash[b] Sample 1 CDD/F conc. (ppb)	Sample 1 TCDD eqts. (ppb) A[c]	B[c]	D[c]	Sample 2 CDD/F conc. (ppb)	Sample 2 TCDD eqts. (ppb) A[c]	B[c]	D[c]
Total PeCDFs	0.1	1	670	67				549	54.9			17.8	1.8		
2378 PeCDFs	0.1	0.07	670		4.7	35.8		549		3.8		17.8		0.1	
other PeCDFs	0.001	0.93	670		0.6	0.3		549		0.5		17.8		--	
Total HxCDFs	0.01	1	965	9.7				1082	10.8			32.1	0.3		
2378 HxCDFs	0.01	0.25	965		2.4	6.7		1082		2.7		32.1		0.1	
other HxCDFs	0.0001	0.75	965		0.1	--		1082		0.1		32.1		--	
Total HpCDFs	0.001	1	460	0.5				499	0.5			10.9		--	
2378 HpCDFs	0.001	0.50	460		0.2	0.3		499		0.2		10.9		--	
other HpCDFs	0.00001	0.50	460		--	--		499		--		10.9		--	
Total estimated TCDD equivalents (TEF)				84	8	46			294	26			9	1	
Measured TCDD Equivalents															
AHH bioassay							--				4				--
EROD bioassay							--				5				--
Receptor binding assay							--				32				4
Acute toxicity bioassay							58				--				--

Table 5. *(continued)*

Isomer	TEF	Propn. factor	MSW fly ash[b] Sample 3 CDD/F conc. (ppb)	Sample 3 TCDD eqts. (ppb) A[c]	Sample 3 TCDD eqts. (ppb) B[c]	Sample 3 TCDD eqts. (ppb) D[c]	Sample 4 CDD/F conc. (ppb)	Sample 4 TCDD eqts. (ppb) A[c]	Sample 4 TCDD eqts. (ppb) B[c]	Sample 4 TCDD eqts. (ppb) D[c]
Total TCDDs	1	1	12.9	12.9			2.4	2.4		
2378 TCDDs	1	0.05	12.9		0.6		2.4		0.1	
other TCDDs	0.01	0.95	12.9		0.1		2.4		--	
Total PeCDDs	0.5	1	37.5	18.8			7.9	4.0		
2378 PeCDDs	0.5	0.07	37.5		1.3		7.9		0.3	
other PeCDDs	0.005	0.93	37.5		0.2		7.9		--	
Total HxCDDs	0.04	1	75	3			9.7	0.4		
2378 HxCDDs	0.04	0.3	75		0.9		9.7		0.1	
other HxCDDs	0.0004	0.7	75		--		9.7		--	
Total HpCDDs	0.001	1	41.9	--			9.1	--		
2378 HpCDDs	0.001	0.5	41.9		--		9.1		--	
other HpCDDs	0.00001	0.5	41.9		--		9.1		--	
Total TCDFs	0.1	1	8.2	0.8			4.4	0.4		
2378 TCDFs	0.1	0.03	8.2		--		4.4		--	
other TCDFs	0.001	0.97	8.2		--		4.4		--	
Total PeCDFs	0.1	1	19.8	2.0			21.0	2.1		
2378 PeCDFs	0.1	0.07	19.8		0.1		21.0		0.1	
other PeCDFs	0.001	0.93	19.8		--		21.0		--	

Table 5. (continued)

Isomer	TEF	Propn. factor	MSW fly ash[b] Sample 3 CDD/F conc. (ppb)	Sample 3 TCDD eqts. (ppb) A[c]	Sample 3 TCDD eqts. (ppb) B[c]	Sample 3 TCDD eqts. (ppb) D[c]	Sample 4 CDD/F conc. (ppb)	Sample 4 TCDD eqts. (ppb) A[c]	Sample 4 TCDD eqts. (ppb) B[c]	Sample 4 TCDD eqts. (ppb) D[c]
Total HxCDFs	0.01	1	38.7	0.4			21.6	0.2		
2378 HxCDFs	0.01	0.25	38.7		0.1		21.6		0.1	
other HxCDFs	0.0001	0.75	38.7		--		21.6		--	
Total HpCDFs	0.001	1	20.6	--			16.6	--		
2378 HpCDFs	0.001	0.50	20.6		--		16.6		--	
other HpCDFs	0.00001	0.50	20.6		--		16.6		--	
Total estimated TCDD equivalents (TEF)				38	2			9	0.7	
Measured TCDD Equivalents										
AHH bioassay						4				2
EROD bioassay						5				2
Receptor binding assay						65				11
Acute toxicity bioassay						--				--

[a]Des Rosiers, 1984, assuming only homologue-specific concentrations are known (for isomer-specific analyses; see Table 4).
[b]Sawyer et al., 1983.
[c]A = estimated assuming 2378-isomers constitute 100% of a homologous group.
B = estimated assuming occurrence of all isomers in a homologous group is equally probable (thus using the proportionality factor in column three).
C = estimated by utilizing isomer-specific analyses (see Table 4).
D = estimated by direct bioassay.
[d]Values rounding off to less than 0.1 are omitted.

limit of the excess risk of developing cancer (from inhalation exposure alone) for a person living downwind from the facility emitting the mixture under consideration, assuming lifetime exposure:

upper 95% limit of excess cancer risk resulting from inhalation exposure
= [potency] × [exposure]
= [1.6×10^5 per mg 2,3,7,8-TCDD/kg-day]
× [32 pg TCDD/mg mixture × 10^{-9} mg 2,3,7,8-TCDD/pg
× 1 ng mixture/kg-day × 10^{-6} mg mixture/ng mixture].

VI. Comparison of the TEF Approach with Results of Biological Testing

A limited number of *in vivo* and *in vitro* approaches have been employed in assessing the toxicity of complex mixtures of CDDs and CDFs. While the results from these attempts are not definitive, it is instructive to compare those results with the results from the TEF approach proposed here.

Eadon et al. (1982) investigated the toxicity of CDD/CDF-contaminated soot associated with a fire involving PCB-containing electrical equipment. Using the results from acute *in vivo* toxicity (LD_{50}) studies in which the soot was the test substance, the researchers determined that it had the acute toxicity expected of material containing about 60 times the amount of 2,3,7,8-TCDD actually found by GC/MS analysis.

Table 5 illustrates the results of employing the TEF approach through three different procedures, each of which depends upon the results of GC/MS analysis of the soot. In the first instance (A, in Table 5), the analytical data have been consolidated to totals within a homologous class. These concentrations are treated as if they consisted completely of 2378-members of the class and, therefore, are multiplied by the TEF appropriate for the 2378-members of the class. The resulting estimate of 2378-TCDD equivalents by this procedure is about 80.

In procedure B the assumption is made that the occurrence of each of the congeners in a homologous class is equally probable; e.g., the concentration of 2,3,7,8-TCDD is 1/22 (about 5%) of the concentration of the total TCDDs. This approach leads to an estimate of the total 2378-TCDD equivalents of 8.

A rather unique data base exists in the case of the soot from this fire in that an extensive isomer-specific analysis of the sample is available (as cited in Des Rosiers, 1984). Therefore, the full array of TEFs from Table 1 (using the current EPA recommendations) can be applied. This procedure (C in Table 5) results in an estimate of roughly 50 for the total 2378-TCDD equivalents in the sample.

As might be expected, the most conservative of these procedures, A, leads to the highest estimate. Approach B (using theoretical probability of occurrence) leads to an estimate that is about 10-fold lower than the isomer-specific results C, reflecting the fact that the 2378-congeners are present in somewhat higher than "equal probability" proportions in this particular soot sample. Given the complexity of the analysis involved, the approximate nature of the TEF method, and the vagaries of the assay, a major feature of note in Table 5 regarding the soot samples is that the results of procedures A, B, and C span a range of only one order of magnitude and bracket the bioassay estimate, reported by Eadon et al. (1982).

Table 5 also shows the results of the application of approaches A and B to published results of homologue-specific CDD and CDF concentrations in fly ash from four municipal solid waste combustors (Sawyer et al., 1983). In addition, extracts from the fly ash samples were analyzed by three bioassay techniques (AHH induction, EROD induction, and receptor binding). Again, the calculated results span an order of magnitude, with the bioassay results lying within or close to this range.

These data suggest that the TEF approach is likely to be a useful interim tool for the rough (order of magnitude) estimation of the toxicity of complex mixtures of CDDs and CDFs. The availability of additional data comparing

the results of analytical and biological assays will enable a conclusion regarding the preferred method of estimating TEFs (e.g., method A or B of Table 5).

VII. Research Needs

The Forum recommends that the Agency support research that would allow actual measurement of mixtures containing CDDs and CDFs, rather than drawing inferences from component toxicity. The results of this research could reduce the need for the TEF approach. In addition, research should be conducted in order to provide a firmer basis for, and to guide appropriate modification of, the TEF approach. Several areas of research are appropriate for these purposes.

1. Validation and completion of the *in vitro* test data such as those listed in Table 2.
2. Investigation of the relationships between short-term *in vivo* and *in vitro* tests and the toxic end points of concern; i.e., carcinogenicity, reproductive toxicity, immunotoxicity, and other singificant human health effects resulting from CDD/CDF exposure.
3. Determination of the impact of pharmacodynamics, including bioavailability, potential for absorption, and toxic potencies of metabolites of CDDs and CDFs in *in vitro* tests, relative to the potencies of the parent compounds. As pointed out by several reviewers, this would enable refinement of the TEF approach.
4. Investigation of additional short-term assays which can test the mechanistic hypotheses underlying the TEF approach.

References

Bandiera, S.; et al. (1983) Competitive binding of the cytosolic tetrachlorodibenzo-*p*-dioxin receptor. Biochem. Pharmacol. 32:3803-3813.

Bandiera, S.; et al. (1984) Polychlorinated dibenzofurans (PCDFs): effects of structure on binding to the 2,3,7,8-TCDD cytosolic receptor protein, AHH induction and toxicity. Toxicology 32:131-144.

Bradlaw, J.; et al. (1979) Induction of enzyme activity in cell culture: a rapid screen for detection of planar polychlorinated organic compounds. J. Assoc. Off. Anal. Chem. 62:904-916.

Bradlaw, J.; et al. (1980) Comparative induction of aryl hydrocarbon hydroxylase activity *in vitro* by analogues of dibenzo-*p*-dioxin. Cosmet. Toxicol. 18:627-635.

Commoner, B.; et al. (1984, May 1) Environmental and economic analysis of alternative municipal solid waste disposal technologies. I. An assessment of the risks due to emissions of chlorinated dioxins and dibenzofurans from proposed New York City incinerators.

Cooper Engineers. (1984) Air emissions and performance testing of a dry scrubber (quench reactor) dry Venturi and fabric filter system operating on flue gas from combusion of municipal solid waste at Tsushima, Japan.

Czuwa, J.M.; Hites, R. (1984) Environmental fate of combustion-generated polychlorinated dioxins and furans. Environ. Sci. Technol. 16:444-450.

Dencker, L.; et al. (1985) Fetal thymus organ culture as an *in vitro* model for the toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and its congeners. Mol. Pharmacol. 27:133-140.

Denison, M.S.; Wilkinson, C.F. (1985) Identification of the Ah receptor in selected mammalian species and induction of aryl hydrocarbon hydroxylase. Eur. J. Biochem. 14:429-435.

Des Rosiers, P. (1984) PCBs, PCDFs, and PCDDs resulting from transformer/capacitor fires: an overview. Proc. 1983 PCB seminar. Research project 2028, Electric Power Research Institute. Palo Alto, California.

Eadon, G.; et al. (1982) Comparisons of chemical and biological data on soot samples from the Binghamton State Office Building. (Unpublished report).

Gasiewicz, T.A.; Rucci, G. (1984) Cytosolic receptor for tetrachlorodibenzo-*p*-dioxin. Evidence for a homologous nature among various mammalian species. Mol. Pharmacol. 26:90-98.

Gierthy, J.F.; Crane, D. (1984) Reversible inhibition of *in vitro* epithelial cell proliferation by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. Toxicol. Appl. Pharmacol. 74:91-98.

Gierthy, J.F.; Crane, D. (1985) *In vitro* bioassay for dioxin-like activity based on alterations in epithelial cell proliferation and morphology. Fundam. Appl. Toxicol. 5:754-759.

Grant, D.L. (1977) Proc. 12th annual workshop on pesticide residues analysis. Winnipeg, Canada. p. 251.

Gravitz, N.; et al. (1983, Nov. 1) Interim guidelines for acceptable exposure levels in office settings contaminated with PCB and PCB combustion products. Epidemiological Studies Section, California Department of Health Services.

Greenlee, W.F.; et al. (1985a) Evidence for direct action of tetrachlorodibenzo-*p*-dioxin on thymus epithelium. Toxicol. Appl. Pharmacol. 79:112-120.

Greenlee, W.F.; et al. (1985b) Toxicology of chlorinated aromatic hydrocarbons in animals and humans: *in vitro* approach to toxic mechanisms. Environ. Health Perspect. 60:69-76.

Greig, J.B.; et al. (1984) Incomplete correlation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin hepatotoxicity with Ah phenotype in mice. Toxicol. Appl. Pharmacol. 74:17-25.

Hassoun, E.; et al. (1984) Teratogenicity of 2,3,7,8-tetrachlorodibenzofuran in the mouse. J. Toxicol. Environ. Health 14:337-351.

Knutson, J.; Poland, A. (1980) Keratinization of mouse teratoma cell line XB produced by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin: an *in vitro* model of toxicity. Cell 22:27-36.

Kociba, R.J.; Cabey, O. (1985) Comparative toxicity and biologic activity of chlorinated dibenzo-*p*-dioxins and furans relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). Chemosphere 14:649-660.

Lamparski, L.L.; et al. (1984) Presence of chlorodibenzo-*p*-dioxins in a sealed 1933 sample of dried municipal sewage sludge. Chemosphere 13:361-365.

Madhukar, B.V.; et al. (1984) Effects of *in vivo* administered 2,3,7,8-tetrachlorodibenzo-*p*-dioxin on receptor binding of epidermal growth factor in the hepatic plasma membrane of rat, guinea pig, mouse, and hamster. Proc. Natl. Acad. Sci. USA 81:7407-7411.

McKinney, J.; McConnell, E. (1982) Structural specificity and the dioxin receptor. Perg. Ser. Environ. Sci. 5:367-381.

Moore, J.A.; et al. (1979) Comparative toxicity of three halogenated dibenzofurans in guinea pigs, mice, and rhesus monkeys. Ann. N.Y. Acad. Sci. 320:151-163.

Murray, F.J.; et al. (1979) Three-generation reproduction study of rats given 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in the diet. Toxicol. Appl. Pharmacol. 50:241-252.

Nagayama, J.; et al. (1985a) Inducing potency of aryl hydrocarbon hydroxylase in human lymphoblastoid cells and mice by polychlorinated dibenzofurans. Environ. Health Perspect. 59:107-112.

Nagayama, J.; et al. (1985b) Genetically mediated induction of aryl hydrocarbon hydroxylase activity in human lymphoblastoid cells by polychlorinated dibenzofuran isomers and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. Arch. Toxicol. 56:230-235.

Neal, R.A.; et al. (1982) The toxicokinetics of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in mammalian systems. Drug Metab. Rev. 13:355-385.

Neal, R.A. (1985) Mechanisms of the biological effects of PCBs, polychlorinated dibenzo-*p*-dioxins, and polychlorinated dibenzofurans in experimental animals. Environ. Health Perspect. 60:41-46.

Okey, A.B.; Vella, L.M. (1984) Elevated binding of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and 3-methylcholanthrene to the Ah receptor in hepatic cytosols from phenobarbital-treated rats and mice. Biochem. Pharmacol. 33:531-538.

Okey, A.B.; et al. (1984) Ah receptor in primate liver: binding of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and carcinogenic aromatic hydrocarbons. Can. J. Physiol. Pharmacol. 62:1292-1295.

Olie, K.; et al. (1983) Formation and fate of PCDD and PCDF from combustion processes. Chemosphere 12:627-636.

Ontario Government. (1982, Dec. 16) Chlorinated dioxins and chlorinated dibenzofurans. Ambient air guideline. Health Studies Service, Ministry of Labour.

Ontario Government. (1984, Dec.) Scientific criteria document for standard development. Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). Ministry of the Environment. No. 4-84. December.

Poland, A.; et al. (1976) 3,4,3',4'-tetrachloroazoxybenzene and azobenzene: potent inducers of aryl hydrocarbon hydroxylase. Science 194:627-630.

Poland, A.; et al. (1979) Mechanism of action of dioxins. Ann. N.Y. Acad. Sci. 320:214-230.

Poland, A.; Knutson, J.C. (1982) 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and related halogenated aromatic hydrocarbons: an examination of the mechanism of toxicity. Annu. Rev. Pharmacol. Toxicol. 22:517-554.

Poland, A.; et al. (1985) Studies on the mechanism of action of halogenated aromatic hydrocarbons. Clin. Physiol. Biochem. 3:147-155.

Rappe, C. (1984) Analysis of polychlorinated dioxins and furans. Environ. Sci. Technol. 18:78A-90A.

Rizzardini, M.; et al. (1983) Toxicological evaluation of urban waste incinerator emissions. Chemosphere 12:559-564.

Safe, S.; et al. (1985) Polychlorinated dibenzofurans: quantitative structure-activity relationships. Chemosphere 14:675-684.

Sawyer, T.; et al. (1983) Bioanalysis of polychlorinated dibenzofuran and dibenzo-*p*-dioxin mixtures in fly ash. Chemosphere 12:529-534.

Schwetz, B.A.; et al. (1973) Toxicity of chlorinated dibenzo-*p*-dioxins. Environ. Health Perspect. 5:87-89.

Swiss Government (Bundesamt fur Umweltschutz, Bern). (1982) Environmental pollution due to dioxins and furans from chemical rubbish incineration plants. Schriftenreighe Umweltschutz. No. 5.

Tong, H.Y.; et al. (1984) Identification of organic compounds obtained from incineration of municipal waste by HPLC and GC/MS. J. Chromatogr. 285:423-441.

U.S. Department of Health and Human Services. (1983, Apr. 29) Levels of concern for hexa- (HCDD), hepta- (HpCDD) and octachlorodibenzo-p-dioxins (OCDD) in chickens and eggs. Memorandum.

U.S. Environmental Protection Agency. (1981, Nov. 19) Interim evaluation of health risks associated with emissions of tetrachlorinated dioxins from municipal waste resource recovery facilities. Office of the Administrator.

U.S. Environmental Protection Agency. (1982) PCB disposal by thermal destruction. National Technical Information Service, Springfield, VA. PB 82-241860.

U.S. Environmental Protection Agency. (1984a, Feb.) Ambient water quality criteria for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. EPA-440/5-84-007.

U.S. Environmental Protection Agency. (1984b, June) Assessment of emissions of specific compounds from a resource recovery municipal refuse incinerator. Office of Toxic Substances. EPA-560/5-84-002.

U.S. Environmental Protection Agency. (1984c, Dec.) Thermal degradation products from dielectric fluids. EPA-560/5-84-009.

U.S. Environmental Protection Agency. (1985a, Jan. 9) Proposed guidelines for the health risk assessment of chemical mixtures and request for comments: notice. Federal Register 50:1170-1176.

U.S. Environmental Protection Agency. (1985b) Health assessment document for polychlorinated dibenzo-*p*-dioxins. EPA-600/8-84-014F. National Technical Information Service, Springfield, VA. PB 86-122546/AS.

Vickers, A.E.M.; et al. (1985) Mechanism of action of toxic halogenated aromatics. Environ. Health Perspect. 59:121-129.

Weber, H.; et al. (1984) Teratogenicity of 2,3,7,8-tetrachlorodibenzofuran (TCDF) in mice. Toxicol. Lett. 20:183-188.

Appendix A
Nomenclature

The following terminology and abbreviations are used in this document:

1. The term "congener" refers to any one particular member of the same chemical family; e.g., there are 75 congeners of chlorinated dibenzo-*p*-dioxins.
2. The term "homologue" refers to a group of structurally related chemicals that have the same degree of chlorination. For example, there are eight homologues of CDDs, monochlorinated through octochlorinated.
3. The term "isomer" refers to substances that belong to the same homologous class. For example, there are 22 isomers that constitute the homologues of TCDDs.
4. A specific congener is denoted by unique chemical notation. For example, 2,4,8,9-tetrachlorodibenzofuran is referred to as 2,4,8,9-TCDF.
5. Notation for homologous classes is as follows:

Dibenzo-*p*-dioxin	D
Dibenzofuran	F

No. of halogens	Acronym	Example
1	M	
2	D	2,4-DCDD
3	Tr	
4	T	1,4,7,8-TCDD
5	Pe	
6	Hx	
7	Hp	
8	O	
1 through 8	CDDs and CDFs	

6. Dibenzo-*p*-dioxins and dibenzofurans that are chlorinated at the 2,3,7, and 8 positions are denoted as "2378" congeners, except when 2,3,7,8-TCDD is uniquely referred to; e.g., 1,2,3,7,8-PeCDF and 2,3,4,7,8-PeCDF are both referred to as "2378-PeCDFs."

Appendix B
Comparison of Different Approaches to Calculating 2378-TCDD Equivalents

Table 1 in the text lists a number of different approaches for calculating 2378-TCDD toxicity equivalents. Five of the approaches (those that deal with 4-position 2378-substituted congeners, but not 3-position substituted congeners) were applied to the data in Table 4 in the text.

These approaches were also applied to some of the data included in Table I of the Report of the Citizens Advisory Committee on Resource Recovery in Brooklyn (March, 1985), produced by Ketcham and the Mt. Sinai School of Medicine.

A summary comparison of the relative results is found in Table B-1, with the supporting tables (Tables B-2 through B-13) attached. (Note that the units of mass emission are not the same for all of the facilities. Therefore, comparison of absolute numbers between facilities may be invalid).

These data indicate that, in general, the methods used by the Swiss government, New York State, and the U.S. EPA (the 1981 approach and the 1985 proposal) all generate results which are within an order of magnitude of each other. This suggests that, within the range considered, the results are not particularly sensitive functions of the TEFs selected.

The procedure recommended by the state of California, however, gives results which are roughly an order of magnitude higher than those generated by the other approaches. In general, the greater the contribution from the TCDDs, the greater the similarity in the results of the methods. This is due to the fact that all methods assign a TEF of 1 for 2,3,7,8-TCDD (and 1 to all TCDDs, when isomer-specific analyses are not available). Because higher chlorinated CDDs and CDFs contribute significantly to the total, the disparity is greater between the state of California results and those produced by the other methods, since California assumes that all 2378-substituted CDDs and CDFs are as potent as 2,3,7,8-TCDD. The other methods acknowledge, to one degree or another, the reduced toxicity of higher chlorinated species; see Table 2.

Table B-1. Relative 2378-TCDD Equivalents[a]

Source	EPA '85	EPA '81	Swiss	NY	CA
St. Louis air particulates	1	0.3	1	2	40
PCB fire soot (isomer-specific)	1	0.03	4	3	30
MSW ESP dust	1	0.2	3	2	30
Lake sediment	1	--	2	2	30
Milorganite	1	0.6	2	0.9	30
Oslo MSW flyash	1	--	1	2	20
Ontario MSW flyash	1	0.8	1	2	3
Japanese plant A	1	0.3	1	2	7
Japanese plant B	1	0.6	0.8	2	3
Albany	1	0.3	0.4	2	5
Wright-Patterson (best)	1	0.2	2	3	20
Wright-Patterson (worst)	1	0.4	2	2	20

[a]Calculated using the Toxicity Equivalence Factors shown in Table 1.

Table B-2. Calculation of 2378-TCDD Toxicity Equivalents for St. Louis Air Particulates Using Homologue-Specific Data

Compound	CDD/F conc. (ppb)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	0.2	1	0.2	1	0.2	1	0.2	1	0.2	1	0.2
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	1	0.5	0.5	0	0	0.1	0.1	1	1	1	1
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	1.2	0.04	0.048	0	0	0.1	0.12	0.03	0.036	1	1.2
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0
2378-HpCDD	25	0.001	0.025	0	0	0.01	0.25	0	0	1	25
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	170	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	NA	0.1	0	0	0	0.1	0	0.33	0	1	0
TCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	NA	0.1	0	0	0	0.1	0	0.33	0	1	0
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	NA	0.01	0	0	0	0.1	0	0.01	0	1	0
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0

Table B-2. *(continued)*

Compound	CDD/F conc. (ppb)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)
2378-HpCDF	NA	0.001	0	0	0	0.1	0	0	0	1	0
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	NA	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			0.7		0.2		0.7		1.2		27.4

Table B-3. **Calculation of 2378-TCDD Toxicity Equivalents for PCB Fire Soot Using Isomer-Specific Data**

Compound	CDD/F conc. (ppm)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppm)	TEFs	TEs (ppm)	TEFs	TEs (ppm)	TEFs	TEs (ppm)	TEFs	TEs (ppm)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	0.6	1	0.6	1	0.6	1	0.6	1	0.6	1	0.6
TCDDs	0.6	0.01	0.006	1	0.6	0.01	0.006	0	0	0	0
2378-PeCDD	2.5	0.5	1.25	0	0	0.1	0.25	1	2.5	1	2.5
PeCDDs	2.5	0.005	0.0125	0	0	0.1	0.25	0	0	0	0

Table B-3. *(continued)*

Compound	CDD/F conc. (ppm)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppm)	TEFs	TEs (ppm)	TEFs	TEs (ppm)	TEFs	TEs (ppm)	TEFs	TEs (ppm)
2378-HxCDD	1.1	0.04	0.044	0	0	0.1	0.11	0.03	0.033	1	1.1
HxCDDs	3.6	0.0004	0.00144	0	0	0.1	0.36	0	0	0	0
2378-HpCDD	3	0.001	0.003	0	0	0.01	0.03	0	0	1	3
HpCDDs	4	0.00001	0.00004	0	0	0.01	0.04	0	0	0	0
OCDD	2	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	12	0.1	1.2	0	0	0.1	1.2	0.33	3.96	1	12
TCDFs	16	0.001	0.016	0	0	0.1	1.6	0	0	0	0
2378-PeCDF	358	0.1	35.8	0	0	0.1	35.8	0.33	118.14	1	358
PeCDFs	312	0.001	0.312	0	0	0.1	31.2	0	0	0	0
2378-HxCDF	670	0.01	6.7	0	0	0.1	67	0.01	6.7	1	670
HxCDFs	295	0.0001	0.0295	0	0	0.1	29.5	0	0	0	0
2378-HpCDF	285	0.001	0.285	0	0	0.1	28.5	0	0	1	285
HpCDFs	172	0.00001	0.00172	0	0	0	0	0	0	0	0
OCDF	40	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			46		1.2		196		132		1332

Table B-4. Calculation of 2378-TCDD Toxicity Equivalents for MSW ESP Dust Using Homologue-Specific Data and 2378 TEFs

Compound	CDD/F conc. (ppb)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	5	1	5	1	5	1	5	1	5	1	5
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	10	0.5	5	0	0	0.1	1	1	10	1	10
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	160	0.04	6.4	0	0	0.1	16	0.03	4.8	1	160
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0
2378-HpCDD	120	0.001	0.12	0	0	0.01	1.2	0	0	1	120
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	260	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	40	0.1	4	0	0	0.1	4	0.33	13.2	1	40
TCDFs	0	.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	80	0.1	8	0	0	0.1	8	0.33	26.4	1	80
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	280	0.01	2.8	0	0	0.1	28	0.01	2.8	1	280
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0

Table B-4. *(continued)*

Compound	CDD/F conc. (ppb)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)
2378-HpCDF	160	0.001	0.16	0	0	0.1	16	0	0	1	160
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	40	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			31		5		79		62		855

Table B-5. Calculation of 2378-TCDD Toxicity Equivalents for Lake Sediment Using Homologue-Specific Data

Compound	CDD/F conc. (ppb)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	0	1	0	1	0	1	0	1	0	1	0
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	0.1	0.5	0.05	0	0	0.1	0.01	1	0.1	1	0.1
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	0.34	0.04	0.0136	0	0	0.1	0.034	0.03	0.0102	1	0.34
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0

Table B-5. *(continued)*

Compound	CDD/F conc. (ppb)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)	TEFs	TEs (ppb)
2378-HpCDD	0.5	0.001	0.0005	0	0	0.01	0.005	0	0	1	0.5
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	1.3	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	0.13	0.1	0.013	0	0	0.1	0.013	0.33	0.0429	1	0.13
TCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	0.14	0.1	0.014	0	0	0.1	0.014	0.33	0.0462	1	0.14
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	0.38	0.01	0.0038	0	0	0.1	0.038	0.01	0.0038	1	0.38
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0
2378-HpCDF	1.13	0.001	0.00113	0	0	0.1	0.113	0	0	1	1.13
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	0.14	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			0.1		0		1.2		0.2		2.7

Table B-6. Calculation of 2378-TCDD Toxicity Equivalents for Milorganite Using Homologue-Specific Data

Compound	CDD/F conc. (ppt)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	206	1	206	1	206	1	206	1	206	1	206
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	0	0.5	0	0	0	0.1	0	1	0	1	0
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	2768	0.04	110.72	0	0	0.1	276.8	0.03	83.04	1	2768
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0
2378-HpCDD	7600	0.001	7.6	0	0	0.01	76	0	0	1	7600
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	60000	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	NA	0.1	0	0	0	0.1	0	0.33	0	1	0
TCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	NA	0.1	0	0	0	0.1	0	0.33	0	1	0
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	NA	0.01	0	0	0	0.1	0	0.01	0	1	0
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0

Table B-6. *(continued)*

Compound	CDD/F conc. (ppt)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)
2378-HpCDF	NA	0.001	0	0	0	0.1	0	0	0	1	0
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	NA	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			324		206		559		289		10600

Table B-7. Calculation of 2378-TCDD Toxicity Equivalents for Oslo MSW Fly Ash Using Homologue-Specific Data

Compound	CDD/F conc. (ppt)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	NA	1	0	1	0	1	0	1	0	1	0
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	11	0.5	5.5	0	0	0.1	1.1	1	11	1	11
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	51	0.04	2.04	0	0	0.1	5.1	0.03	1.53	1	51
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0

Table B-7. *(continued)*

Compound	CDD/F conc. (ppt)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)
2378-HpCDD	119	0.001	0.119	0	0	0.01	1.19	0	0	1	119
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	186	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	NA	0.1	0	0	0	0.1	0	0.33	0	1	0
TCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	NA	0.1	0	0	0	0.1	0	0.33	0	1	0
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	NA	0.01	0	0	0	0.1	0	0.01	0	1	0
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0
2378-HpCDF	NA	0.001	0	0	0	0.1	0	0	0	1	0
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	NA	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			7.7		0		7.4		12.5		181

Table B-8. Calculation of 2378-TCDD Toxicity Equivalents for Ontario MSW Fly Ash Using Homologue-Specific Data

Compound	CDD/F conc. (ppt)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)	TEFs	TEs (ppt)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	541	1	541	1	541	1	541	1	541	1	541
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	467	0.5	233.5	0	0	0.1	46.7	1	467	1	467
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	591	0.04	23.64	0	0	0.1	59.1	0.03	17.73	1	591
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0
2378-HpCDD	434	0.001	0.434	0	0	0.01	4.34	0	0	1	434
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	467	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	NA	0.1	0	0	0	0.1	0	0.33	0	1	0
TCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	NA	0.1	0	0	0	0.1	0	0.33	0	1	0
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	NA	0.01	0	0	0	0.1	0	0.01	0	1	0
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0

Table B-8. *(continued)*

Compound	CDD/F conc. (ppt)	EPA 1985 TEFs	EPA 1985 TEs (ppt)	EPA 1981 TEFs	EPA 1981 TEs (ppt)	Switzerland TEFs	Switzerland TEs (ppt)	New York TEFs	New York TEs (ppt)	California TEFs	California TEs (ppt)
2378-HpCDF	NA	0.001	0	0	0	0.1	0	0	0	1	0
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	NA	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			799		541		651		1026		2033

Table B-9. Calculation of 2378-TCDD Toxicity Equivalents for MSW at Japanese Plant A Using Homologue-Specific Data

Compound	CDD/F conc.[a]	EPA 1985 TEFs	EPA 1985 TEs[a]	EPA 1981 TEFs	EPA 1981 TEs[a]	Switzerland TEFs	Switzerland TEs[a]	New York TEFs	New York TEs[a]	California TEFs	California TEs[a]
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	0.1	1	0.1	1	0.1	1	0.1	1	0.1	1	0.1
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	0.07	0.5	0.035	0	0	0.1	0.007	1	0.07	1	0.07
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	0.04	0.04	0.0016	0	0	0.1	0.004	0.03	0.0012	1	0.04
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0

Table B-9. (continued)

Compound	CDD/F conc.[a]	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs[a]	TEFs	TEs[a]	TEFs	TEs[a]	TEFs	TEs[a]	TEFs	TEs[a]
2378-HpCDD	0.02	0.001	0.00002	0	0	0.01	0.0002	0	0	1	0.02
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	0.01	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	1.31	0.1	0.131	0	0	0.1	0.131	0.33	0.4323	1	1.31
TCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	0.38	0.1	0.038	0	0	0.1	0.038	0.33	0.1254	1	0.38
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	0.06	0.01	0.0006	0	0	0.1	0.006	0.01	0.0006	1	0.06
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0
2378-HpCDF	0.01	0.001	0.00001	0	0	0.1	0.001	0	0	1	0.01
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	0.004	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			0.3		0.1		0.3		0.7		2.0

[a]Units = lb/MM BTU($\times 10^{-6}$)

Table B-10. Calculation of 2378-TCDD Toxicity Equivalents for MSW at Japanese Plant B Using Homologue-Specific Data

Compound	CDD/F conc.[a]	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs[a]	TEFs	TEs[a]	TEFs	TEs[a]	TEFs	TEs[a]	TEFs	TEs[a]
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	0.58	1	0.58	1	0.58	1	0.58	1	0.58	1	0.58
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	0.47	0.5	0.235	0	0	0.1	0.047	1	0.47	1	0.47
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	0.36	0.04	0.0144	0	0	0.1	0.036	0.03	0.0108	1	0.36
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0
2378-HpCDD	0.08	0.001	0.00008	0	0	0.01	0.0008	0	0	1	0.08
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	0.04	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	1.25	0.1	0.125	0	0	0.1	0.125	0.33	0.4125	1	1.25
TCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	0.46	0.1	0.046	0	0	0.1	0.046	0.33	0.1518	1	0.46
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	0.06	0.01	0.0006	0	0	0.1	0.006	0.01	0.0006	1	0.06
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0

Table B-10. *(continued)*

Compound	CDD/F conc.[a]	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs[a]	TEFs	TEs[a]	TEFs	TEs[a]	TEFs	TEs[a]	TEFs	TEs[a]
2378-HpCDF	0.02	0.001	0.00002	0	0	0.1	0.002	0	0	1	0.02
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	0.01	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			1.0		0.6		0.8		1.6		3.3

[a]Units = lb/MM BTU($\times 10^{-6}$)

Table B-11. Calculation of 2378-TCDD Toxicity Equivalents for MSW at Albany Using Homologue-Specific Data

Compound	CDD/F conc. (ng/m^3)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	0.45	1	0.45	1	0.45	1	0.45	1	0.45	1	0.45
TCDDs	14	0.01	0.14	1	14	0.01	0.14	0	0	0	0
2378-PeCDD	97	0.5	48.5	0	0	0.1	9.7	1	97	1	97
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	53	0.04	2.12	0	0	0.1	5.3	0.03	1.59	1	53
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0

Table B-11. **(continued)**

Compound	CDD/F conc. (ng/m^3)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)
2378-HpCDD	71	0.001	0.071	0	0	0.01	0.71	0	0	1	71
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	10	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	2.1	0.1	0.21	0	0	0.1	0.21	0.33	0.693	1	2.1
TCDFs	33	0.001	0.033	0	0	0.1	3.3	0	0	0	0
2378-PeCDF	21	0.1	2.1	0	0	0.1	2.1	0.33	6.93	1	21
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	4	0.01	0.04	0	0	0.1	0.4	0.01	0.04	1	4
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0
2378-HpCDF	1	0.001	0.001	0	0	0.1	0.1	0	0	1	1
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	2	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			54		14		22		107		250

Table B-12. Calculation of 2378-TCDD Toxicity Equivalents for WP AFB (Best) Using Homologue-Specific Data

Compound	CDD/F conc. (ng/m^3)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	0.4	1	0.4	1	0.4	1	0.4	1	0.4	1	0.4
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	0.4	0.5	0.2	0	0	0.1	0.04	1	0.4	1	0.4
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	1	0.04	0.04	0	0	0.1	0.1	0.03	0.03	1	1
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0
2378-HpCDD	3	0.001	0.003	0	0	0.01	0.03	0	0	1	3
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	3	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	8	0.1	0.8	0	0	0.1	0.8	0.33	2.64	1	8
TCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	3	0.1	0.3	0	0	0.1	0.3	0.33	0.99	1	3
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	4	0.01	0.04	0	0	0.1	0.4	0.01	0.04	1	4
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0

Table B-12. (continued)

Compound	CDD/F conc. (ng/m^3)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)
2378-HpCDF	9	0.001	0.009	0	0	0.1	0.9	0	0	1	9
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	2	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			1.8		0.4		3.0		4.5		28.8

Table B-13. Calculation of 2378-TCDD Toxicity Equivalents for WP AFB (Worst) Using Homologue-Specific Data

Compound	CDD/F conc. (ng/m^3)	EPA 1985		EPA 1981		Switzerland		New York		California	
		TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)	TEFs	TEs (ng/m^3)
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDD	4	1	4	1	4	1	4	1	4	1	4
TCDDs	0	0.01	0	1	0	0.01	0	0	0	0	0
2378-PeCDD	3	0.5	1.5	0	0	0.1	0.3	1	3	1	3
PeCDDs	0	0.005	0	0	0	0.1	0	0	0	0	0
2378-HxCDD	6	0.04	0.24	0	0	0.1	0.6	0.03	0.18	1	6
HxCDDs	0	0.0004	0	0	0	0.1	0	0	0	0	0

Table B-13. *(continued)*

Compound	CDD/F conc. (ng/m^3)	EPA 1985 TEFs	EPA 1985 TEs (ng/m^3)	EPA 1981 TEFs	EPA 1981 TEs (ng/m^3)	Switzerland TEFs	Switzerland TEs (ng/m^3)	New York TEFs	New York TEs (ng/m^3)	California TEFs	California TEs (ng/m^3)
2378-HpCDD	32	0.001	0.032	0	0	0.01	0.32	0	0	1	32
HpCDDs	0	0.00001	0	0	0	0.01	0	0	0	0	0
OCDD	16	0	0	0	0	0	0	0	0	0	0
Mono to tri	x	0	0	0	0	0	0	0	0	0	0
2378-TCDF	31	0.1	3.1	0	0	0.1	3.1	0.33	10.23	1	31
TCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-PeCDF	15	0.1	1.5	0	0	0.1	1.5	0.33	4.95	1	15
PeCDFs	0	0.001	0	0	0	0.1	0	0	0	0	0
2378-HxCDF	23	0.01	0.23	0	0	0.1	2.3	0.01	0.23	1	23
HxCDFs	0	0.0001	0	0	0	0.1	0	0	0	0	0
2378-HpCDF	93	0.001	0.093	0	0	0.1	9.3	0	0	1	93
HpCDFs	0	0.00001	0	0	0	0	0	0	0	0	0
OCDF	8	0	0	0	0	0	0	0	0	0	0
Total 2378-TCDD equivalents			11.0		4		21.4		22.6		207